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Calibration of a new ear oximeter in humans during exposure to centrifugation.

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An opto-electronic ear oximeter (Hewlett-Packard, Model 47201A) was evaluated as a non-invasive method for determining arterial oxygen saturation (Sa_{O_2}) in human subjects during exposure to various levels of accelerative forces. This physiologic calibration involved exposing 5 subjects while breathing air and wearing the ear oximeter to 3 levels of accelerative forces (3, 5, and 7 G) for 60 sec each, while arterial blood samples were withdrawn concurrently. Sa_{O_2} were determined indirectly from the oxygen tensions (Pa_{O_2}) measured from the

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sampled arterial blood with a blood gas analyzer and corrected to a pH of 7.4 and for excess base. These data were compared, as were similar data taken from the same subjects breathing 3 different hypoxic gas mixtures while resting at normal Earth's gravity (1 G). Regression analyses of these data for both experimental groups [(a) G exposure or (b) hypoxic exposure], comparing the ear oximeter Sa_{O_2} with the Sa_{O_2} obtained from the corrected Pa_{O_2}, found the ear oximeter to be accurate with correlation coefficients of 0.95 and 0.98 respectively.

Calibration of a new ear oximeter in humans during exposure to centrifugation

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BESCH, E. L., F. W. BAUMGARDNER, R. R. BURTON, K. K. GILLINGHAM, R. F. MCPHERSON, AND S. D. LEVERETT, JR. *Calibration of a new ear oximeter in humans during exposure to centrifugation*. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 44(3): 483–487, 1978. – An optoelectronic ear oximeter (Hewlett-Packard, model 47201A) was evaluated as a noninvasive method for determining arterial oxygen saturation (Sa_{O_2}) in human subjects during exposure to various levels of accelerative forces. This physiological calibration involved exposing five subjects, while breathing air and wearing the ear oximeter, for 60 s to each of three levels of accelerative forces (3, 5, and 7 G); arterial blood samples were withdrawn concurrently. Sa_{O_2} was calculated indirectly from the oxygen tensions (Pa_{O_2}) measured from the sampled arterial blood with a blood gas analyzer and corrected for pH and base excess. These data were compared, as were similar data taken from the same subjects breathing three different hypoxic gas mixtures while resting at earth's gravity (1 G). Regression analyses of these data for both experimental groups (*a*, G exposure, or *b*, hypoxic exposure), comparing the ear-oximeter Sa_{O_2} with the calculated Sa_{O_2}, showed the ear oximeter to be accurate with correlation coefficients of 0.95 and 0.98, respectively.

arterial oxygenation; blood oxygen; human acceleration

THE RECENT ADVENT of high-performance fighter aircraft capable of routinely attaining high acceleration forces (termed G)[1] for prolonged durations (>6 G for 15 s) has necessitated human experimentation into this "new" dynamic environment of high sustained G (HSG) (2). Human exposure to accelerative forces of these magnitudes and durations is known to significantly reduce arterial oxygen tensions and saturations (2, 4, 11). To ensure safety of experimental subjects during exposures to HSG, it is important that critical physiological parameters be continuously monitored, i.e., heart rate, heart rhythm, and at times, arterial blood oxygen saturation.

The use of a photoelectric pickup device to provide a continuous indication of the oxygen saturation of circulating arterial blood was first described by Matthes (8). This device, called an "oximeter" by Millikan (9), greatly facilitated studies of arterial oxygen saturation changes in man. Because the early oximeters (5, 8, 9) gave variable results and were difficult to calibrate, design improvements were necessitated to assure that arterial oxygen saturation was not affected by age, race, sex, or blood hemoglobin content of the subject (15). Two excellent and informative reviews of these oximeters and their application are available (10, 16).

The accuracy and practicability of a recently developed optoelectronic ear oximeter (Hewlett-Packard, model 47201A) has been verified (12) for routine clinical use. To validate its value for monitoring arterial oxygen saturation during exposure to HSG, this ear oximeter was calibrated against oxygen tensions of arterial blood samples withdrawn from humans during exposures to various levels of G and hypoxia, and the data are reported herein.

MATERIALS AND METHODS

All G exposures were on a centrifuge of radius 20 ft (6.1 m) located at the USAF School of Aerospace Medicine, Brooks Air Force Base, Texas. Each subject was exposed to 3-, 5-, and 7-G levels of accelerative force for 60 s. Rest periods of several minutes were allowed between each G-level run – the actual length of each rest period varied and was determined by the medical monitor and the subject. Each subject wore the standard anti-G suit (CSU-13 A/P) which was inflated using the standard pressurization schedule of 1.5 (lb/in.^{2})/G beginning at 2 G. All subjects were exposed to G, seated so that their backs were 65° from the accelerative force vector, approaching a supine position. At this back angle, approximately 68% of the subject's vertical axis is supine ($+G_x$) and 32% is upright ($+G_z$).[2] It has been shown that this position relative to G allows the subject to tolerate more easily these high accelerative forces but does not influence the desaturation phenomenon (1, 14).

Five male volunteer subjects were used in this study and all had been trained to tolerate HSG.[2] Four subjects were nonsmokers. All of the subjects had passed a flight physical examination. Their ages ranged from 20 to 29 yr (mean ±SE, 24 ± 2 yr), and they weighed 163–206 lb (190 ± 7.7 lb).

[1] Details regarding nomenclature used in acceleration physiological research are available (6).

[2] The voluntary informed consent of the subjects used in this research was obtained in accordance with AFR 80-33. For a subject to tolerate these high G levels, he must perform a muscle-tensing respiratory type physical activity called the "M-1 maneuver." This effort requires a considerable amount of muscle/respiration coordination which must be learned. The physiological responses and a description of the M-1 are available (2).

Approximately 1 wk prior to the G exposures, 40 ml of venous blood were taken from each subject. Aliquots were appropriately tonometered at several oxygen tensions; hemoglobin concentration was determined; and oxygen content was determined by the manometric Van Slyke method so that each subject's saturation curve could be constructed. None of these O_2 dissociation curves were displaced from accepted normals.

On the day of his acceleration exposures, each subject was examined by a physician for immediate health problems. The subject was then surgically prepared for a radial artery catheterization. The cannula (Medicut, no. 20) remained in the subject for about 4 h. Prior to acceleration exposures, each subject was allowed to breathe several nitrogen-oxygen mixtures in series (15.0%, 12.0%, and 10.9% O_2, remainder nitrogen) before returning to air. These oxygen concentrations were chosen since the Pa_{O_2}, and therefore arterial oxygen saturations, resulting from breathing these gas mixtures at 1 G would approximate those obtained while breathing air during 60-s exposures to 3, 5, and 7 G.

The following arterial blood samples of 5 ml each were taken *a*) while breathing air at 1 G, *b*) after physiological stabilization to each low-oxygen mixture at 1 G, *c*) immediately preceding each G exposure, *d*) during the last 5 s of each 60-s G exposure, and *e*) post-G exposure at the time blood oxygen saturation (measured by ear oximeter) had returned to pre-G levels. Each sample was drawn anaerobically into a sterile heparinized syringe. A mercury-filled cap was affixed to the syringe, and the sample was placed immediately in an ice bath until Pa_{O_2}, Pa_{CO_2}, and pH determinations were made at 37°C (Instrumentation Laboratory blood gas analyzer model 313), usually within 30 min. Since sampling was repeated for each level of acceleration exposure, a total of approximately 70 ml of blood was withdrawn from each subject on the day of the study.

All blood samples were manually taken except those during the last 5 s of each G exposure. These samples were withdrawn using an automatic blood-withdrawal pump system actuated electronically from the control room and attached to their arterial catheter (7). Pump tubing and cannulas were flushed manually with sterile, heparinized saline between acceleration exposures.

The ear oximeter was attached to a Velcro strap headmount to prevent movement of the instrument during centrifugation (Fig. 1). Prior to placement of the oximeter, the right ear pinna was rubbed vigorously with an alcohol-soaked cotton pledget to promote arterialization in the transilluminated ear tissue. A built-in ear-probe heating element maintained increased blood flow through the ear.

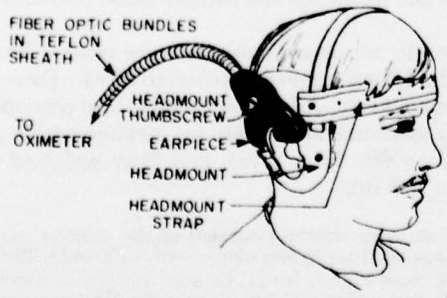

FIG. 1. Ear oximeter probe positioned on ear. Adjustable headband was modified with additional headstraps to prevent shifting of probe during high G exposure.

During all acceleration exposures the subject's heart rate and rhythm were monitored continuously by electrocardiogram (ECG) recordings from sternal and biaxillary leads. Two-way voice contact was maintained with all subjects, and closed-circuit color television was used to monitor them visually during all G exposures.

Analog signals of these physiological parameters were recorded simultaneously on a Brush recorder (Mark 200) and on a Sangamo magnetic tape recorder (model 4742).

RESULTS

A typical ear oximeter recording from one subject during a 60-s exposure to 7 G is shown in Fig. 2. This analog recording is regular and easy to read so that the level of arterial oxygen saturation of the subject is immediately available during G exposure. This type of continuous response recording also allows for later interpretation of the relation between the G environment and various respiratory aspects (3).

Fig. 3 shows the mean values of the ear oximeter readings taken at 10-s intervals of all five subjects exposed for 60 s at each of three levels of G. The oximeter response, both qualitative and quantitative, was similar to that of previous acceleration studies where an ear oximeter and arterial cuvette techniques were used simultaneously during exposures to various $\pm G_x$ levels (11). These investigators reported on eight subjects at $+5.6$ G_x for 3 min and found that the beginning of desaturation occurred 18 s after G onset; as G exposure continued, the change in saturation became less until at approximately 100 s of the 180-s exposure, arterial saturation had essentially reached its minimum value of 86%. This type of desaturation during G was characteristic of the response we found.

More recent work on the effect of sustained G exposure on oxygen desaturation has been reviewed by Burton et al. (2). They developed an equation describing the effect of sustained G on $Sa_{O_2}(\%)$

$$Sa_{O_2} = 99 - 1.59\,G \qquad (1)$$

This equation was developed from Pa_{O_2} values adjusted for changes in Pa_{CO_2} and pH. Arterial saturation values for 3, 5, and 7 G as predicted by *Eq. 1* are compared in Table 1 with values obtained herein using the ear oximeter. The percentage differences between predicted and measured saturation values are small, with a maximum error of 3.5% occurring at 7 G.

Further evaluation of the ear oximeter during G exposure involved comparing oxygen saturations obtained when gas mixtures low in oxygen were inspired at 1 G with saturations found when air was breathed during sustained G exposures. The arterial gas tensions and pH obtained from both methods of developing hypoxemia are compared in Table 2.

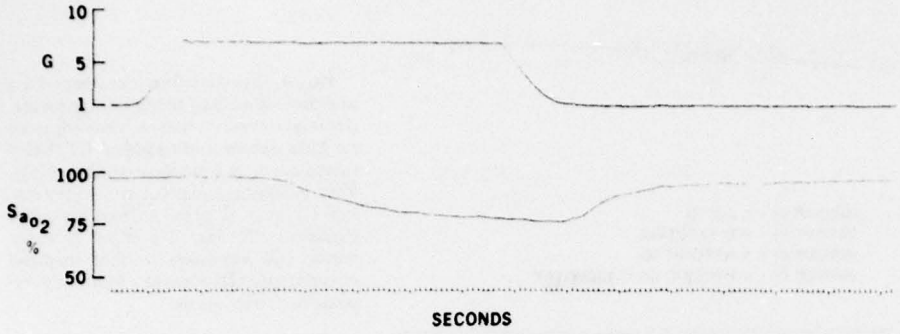

FIG. 2. Typical analog record of the oximeter response duirng 60-s exposure to 7 G.

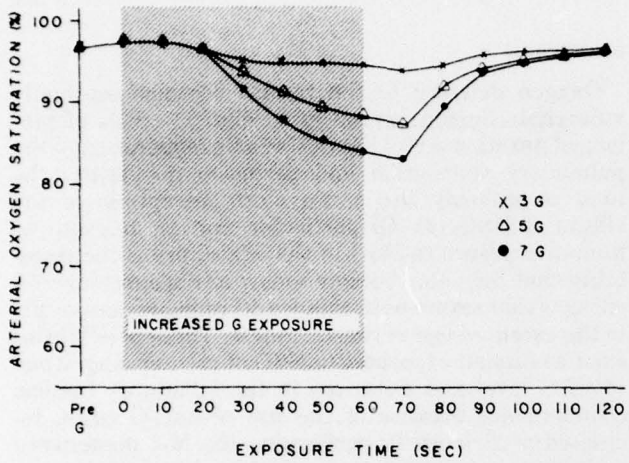

FIG. 3. Change in measured (ear oximeter) arterial oxygen saturation during and after exposure to acceleration. Each point represents mean value of all 5 subjects as exposed to 3, 5, and 7 G. Each G exposure lasted 60 s.

TABLE 1. *Comparison of predicted and measured arterial oxygen saturations for various G levels*

G level	Predicted*	Measured†	% Difference‡
3	94.2	94.7 + 0.82	+0.5
5	91.0	89.8 ± 1.59	−1.3
7	87.8	84.7 ± 2.19	−3.5

* Predicted values based on equation Sa_{O_2} = 99 − 1.59 G (2). † Measured values obtained from ear oximeter (mean ± SE). ‡ % Difference = (measured − predicted)/predicted × 100.

Values for sustained G exposures are similar to those previously reported (2), reflecting significant reductions in Pa_{O_2} with increasing G levels yet a consistently reduced Pa_{CO_2} at all G levels. This low Pa_{CO_2} is a function of the M-1 maneuver (moderate hyperventilation)[2] and probably would be lower if not for the venous admixture to the arterial blood via a right-left pulmonary shunt, which is significant during even low levels of G exposures (4, 14). Clearly the hypoxic inspired gases were sufficiently low in oxygen to reduce the Pa_{O_2} to levels near those obtained during G exposures. Some reduction in Pa_{CO_2}, especially in persons breathing 10.9% oxygen, suggested the occurrence of some degree of hyperventilation.

Oximeter readings are plotted as a function of arterial oxygen tensions (adjusted for base excess and to pH of 7.4 using the equation $\Delta \log P_{O_2} = -0.48\ \Delta pH + 0.0013$ BE) and compared with the standard oxygen saturation curve (13) in Fig. 4. The experimentally obtained data are closely correlated with the standard oxygen saturation curve. Seven Pa_{O_2} values are high (≥110 Torr). These occurred at 1 G prior to a G exposure, except for one which was found at 3 G. These points were associated with a low Pa_{CO_2}, suggesting that the subjects were hyperventilating at that time, a common occurrence prior to G exposure. No consistent differences are found between increased G exposures and the 1 G hypoxic environment, indicating, as reported by Nolan et al. (11) using a earlier model ear oximeter, that changes in blood content in the transilluminated ear tissue and blood hemoglobin concentration due to acceleration apparently do not affect the operation of this more recent model ear oximeter.

TABLE 2. *Measured Pa_{O_2}, Pa_{CO_2}, and pH of 5 subjects as determined for each experimental condition*

Experimental Condition	Pa_{O_2}	Pa_{CO_2}	pH
1 G, air (control)	91.3 ± 3.1	37.9 ± 1.4	7.413 ± 0.004
1 G, 15.0% oxygen	66.6 ± 4.0	35.7 ± 0.6	7.442 ± 0.004
1 G, 12.0% oxygen	51.8 ± 3.2	35.5 ± 0.7	7.456 ± 0.002
1 G, 10.9% oxygen	42.7 ± 3.3	33.8 ± 0.6	7.467 ± 0.004
3 G, air	76.2 ± 7.7	32.7 ± 1.2	7.466 ± 0.013
5 G, air	53.6 ± 2.9	30.0 ± 0.6	7.474 ± 0.011
7 G, air	46.4 ± 3.0	30.3 ± 0.9	7.450 ± 0.019

All values are means ± SE. Values obtained from all experimental conditions significantly differed ($P < 0.05$) from 1 G air-breathing control values based on two-way analysis of variance and Student *t*-test.

The Sa_{O_2} of each blood sample was determined from the Pa_{O_2} measured by the blood gas calculator (13) and was statistically compared to the reading of the ear oximeter, using the correlation coefficient. These comparisons for the hypoxic and G exposures are shown in Fig. 5. The regression analyses for these two groups are

G exposure

$$Sa_{O_2(e)} = 2.7 + 0.97\ Sa_{O_2(co)} \qquad (2)$$

$$r = 0.95;\ P < 0.001$$

Hypoxic

$$Sa_{O_2(e)} = 1.13\ Sa_{O_2(co)} - 12.2 \qquad (3)$$

$$r = 0.98 +\ P < 0.001$$

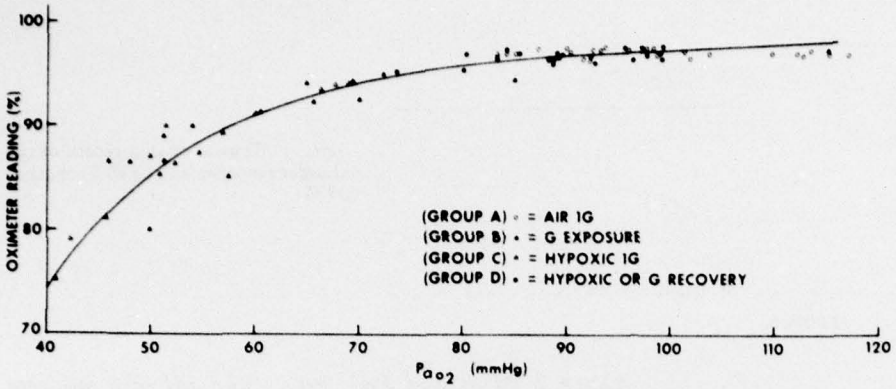

FIG. 4. Relationship between Pa_{O_2} and measured Sa_{O_2} using ear oximeter. Arterial oxygen tensions were adjusted for base excess and to pH of 7.4. Saturation curve is from Severinghaus (13). Four groups are identified: (*A*) breathing air at 1 G prior to hypoxic or G exposure; (*B*) last 5 s of all G exposures; (*C*) exposure to low inspired oxygen; and (*D*) recovery following hypoxic or G exposures.

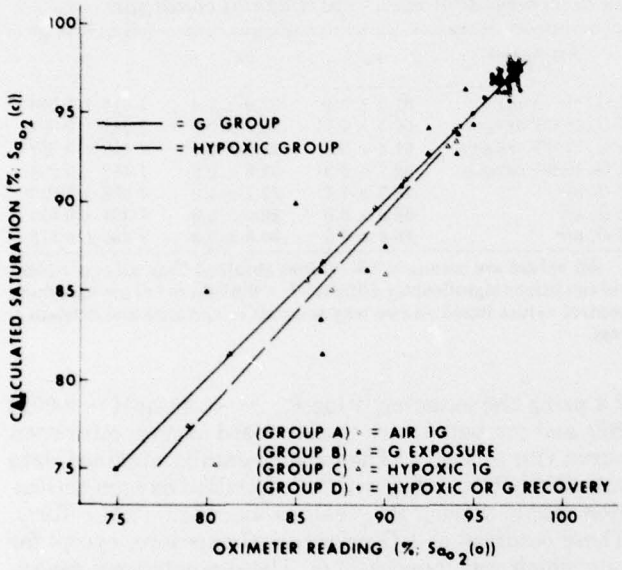

FIG. 5. Individual subject oximeter readings are compared with Sa_{O_2} calculated from arterial blood gas tensions adjusted for base excess and to a pH of 7.4. Four groups are identified (see Fig. 4). Regression lines (solid line for G exposure and broken line for hypoxic exposure) were calculated from *Eqs. 2* and *3* of text.

where for *Eqs. 2* and *3*

$Sa_{O_{2(c)}}$ = arterial saturation determined from adjusted Pa_{O_2}

$Sa_{O_{2(o)}}$ = oximeter reading

The near-perfect correlation coefficients of 0.95 and 0.98 are similar to the correlation coefficient of 0.97 reported by Saunders et al. (12) using the same model ear oximeter while exposing subjects at 1 G to "acute, progressive and steady-state isocapnic hypoxia."

The rapid recovery of arterial saturation at 1 G following exposures to sustained G—essentially complete < 60 s post-G (Figs. 2 and 3)—was verified from arterial blood withdrawn from the subjects at that time. The ear oximeter reading was 96.7 ± 0.15 and the calculated Sa_{O_2} from the arterial blood was 96.8 ± 0.11, not a statistically significant difference between methods of determining the Sa_{O_2}.

DISCUSSION

Oxygen delivery to the tissues becomes especially vulnerable during exposures to high G levels of prolonged duration ≥60 s because of an exaggeration of the pulmonary ventilation and perfusion inequality; the lung is probably the organ most susceptible to the effects of HSG (2). Of particular interest relevant to human exposure to HSG in the laboratory is the possibility that Sa_{O_2} may become so low in the experimental subjects that severe performance decrements may occur, to the extent of loss of consciousness. These possibilities exist as human exposures to HSG of greater magnitude (both G level and duration) in the laboratory become commonplace because of the use of anti-G suits, increased proficiency in performing the M-1 maneuver,[2] and the use of aircraft seats which recline subjects into the supine $+G_x$ mode. All of the methods used to increase G tolerance counteract the effect of G on the cardiovascular system, but without benefit (and possibly some detriment) to the respiratory system; e.g., it appears that both the anti-G suit and $+G_x$ posture may increase the reduction of Sa_{O_2} which occurs during G exposure (1, 4). Even the addition of oxygen to the inspired gases is not without hazard (absorptional atelectasis) and may not be effective in increasing Sa_{O_2} because of the type of pulmonary shunting that occurs during exposure to HSG (4).

Since the respiratory system is significantly affected by HSG, understanding this relationship is important. Some of the respiratory physiology of HSG (especially the kinetics) may be developed using the ear oximeter (3). Such a device is especially useful in the G environment since *a*) it is continuously and rapidly responsive to changes in this dynamic environment; *b*) it is small and light and does not encumber the subject; *c*) being noninvasive, this device is welcomed by the volunteer subject and does not add stress to an already stressed person; and *d*) the data obtained from the subject are immediately transmitted to the investigator, who does not share the environment with the subject. Consequently, it appears that the ear oximeter is destined to become a valuable research tool for acceleration physiologists.

As a medical monitoring device to insure the health of our subjects, we routinely use this ear oximeter during several types of high G exposures, and on

occasion, for specific experimental protocols, its use has been a requirement of our Advisory Committee on Human Experimentation. Also, it has been reliable in the high G environment in that we have used it for a year without a machine failure during approximately 1,700 exposures.

Recent research has suggested that significant decrements in performance are associated with minor reductions in Pa_{O_2} (data unpublished). In the future, considering the potential magnitude of G forces possible with high-performance aircraft and the high level of pilot mental alertness required by the aircraft's technical sophistication, it may become necessary to monitor the Sa_{O_2} of pilots so they can be aware of possible compromise in their performance. Measuring a pilot's Sa_{O_2} may soon be possible with a modification of this ear oximeter or some other type of oxygen-sensing device.

The research reported in this paper was conducted by personnel of the Crew Technology Division, USAF School of Aerospace Medicine, Aerospace Medical Division, AFSC, United States Air Force, Brooks Air Force Base, Texas.

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